The treatment of overactive bladder, detrusor underactivity, and stress incontinence

(Leczenie pęcherza nadreaktywnego, niewydolności skurczowej wypieracza i wysiłkowego nietrzymania moczu)

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Abstract – The authors have paid attention to the changing terminology that defines bladder-related disorders, which stems from the problematic ethiopathogenesis of the ailments. They have discussed selected issues related to bladder and urethra physiology while being filled and emptied as well as sensitivity disorders of the bladder. They have characterised the epidemiology, clinical symptoms, and selected treatment methods of overactive bladder. A subject of another analysis was detrusor underactivity – the causes and some treatment methods have been discussed. The final disorder discussed here is stress incontinence. The authors have emphasised the significance of the ailments that "mask" stress incontinence, and highlighted the significance of diagnostics and treatment.

Key words **-** overactive bladder, detrusor underactivity, stress urinary incontinence, diagnostics, treatment.

Streszczenie – Autorzy zwrócili uwagę na zmieniającą się terminologię określenie zaburzeń czynności pęcherza moczowego co wynika z trudności właściwego ustalenia etiopatogenezy zaburzeń. Omówili wybrane zagadnienia z fizjologii pęcherza moczowego i cewki moczowej w trakcie napełniania i opróżniania oraz zaburzenia czucia pęcherzowego. Następnie scharakteryzowali epidemiologię występowania, objawy kliniczne i wybrane metody leczenia pęcherza nadreaktywnego. Przedmiotem analizy była także niewydolność skurczowa wypieracza. Omówiono przyczyny tej niewydolności oraz niektóre metody leczenia. Kolejnym omówionym zaburzeniem było wysiłkowe nietrzymanie moczu. Autorzy podkreślili rolę schorzeń „maskujących" wysiłkowe nietrzymanie moczu oraz znaczenie diagnostyki i leczenia.

Słowa kluczowe pęcherz nadreaktywny, niewydolność skurczowa wypieracza, wysiłkowe nietrzymanie moczu, diagnostyka, leczenie.

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I. INTRODUCTION – TERMINOLOGY AND PHYSIOLOGY

Over the years, various names were applied to define bladder disorders: spinal, spastic, contractile, automatic, dyssynergic, unstable, or hyperreflectoric. This review of terminology allows one to conclude that, while some terms are based on descriptive attempts to characterise bladder activity, other ones are aimed at reflecting the neuropathologic process lying at the foundations of micturition problems. Contemporary nomenclature has been ordered, but there are still two systems of classification: a neurological and a functional one.

Currently, the recommendations of the ICS (International Continence Society) Standardisation Committee, the functional and descriptive terminology base established in 1988 should be used [1]. It describes the parameters of bladder and urethra while they are being filled and emptied. When it is being filled up, the bladder has to be a stable and susceptive container. All anomalies from that are pathological.

The contractions that take place while bladder is being filled are considered as unstable bladder. When a bladder is small and has rigid walls, its susceptibility (increase in the internal pressure against the increase of volume) is decreased. The reverse – an excessively flexible and elastic bladder – is also an anomaly. It's susceptibility is increased in that case, too [2].

Bladder sensitivity disorders are related to oversensitive receptors (decreased sensitivity threshold) or decreased sensitivity (increased threshold, no sensitivity). The term "unstable detrusor" calls for some explanation. It is a functional diagnosis made only on the basis of a urodynamic study. While it is not an ICS Standardisation Committee benchmark, it has been assumed that instability is a pressure change when the bladder is being filled by more than 15 cm H_2O, regardless of the cause (be I neurological or idiopathic). When the bladder is filled, the urethral functions might be correct or disrupted – in which case the stress incontinence occurs [1,3].

The emptying, or micturition, occurs when the urine leaves the bladder. The detrusor has to shrink as much as possible. However, sometimes the force of the contraction might be decreased, or the contraction might be impossible.

The urine flow through urethra has to occur with the least resistance possible. Therefore, during micturition, the urethra should relax accordingly. However, sometimes there might be obstructions: anatomical ones (such as benign enlargement of the prostate, narrow urethra, or posterior urethral valves) or functional ones (such as detrusor or sphincter disorders, spastic sphincters). This type of descriptive systematics of lower urinary tract functions allows one to define the disorder precisely [3].

On the other hand, it is not always the case that a urodynamic study allows one to credibly and repetitively reproduce a patient's ailments. This pertains predominantly to an "unstable detrusor". This is why Paul Abrams proposed to introduce the term "overactive bladder" in 1997. According to his definition, symptomatic bladder overactivity is a condition when polyuria, urinary urgency, and urgency incontinence are the case – separately, or jointly, with no local pathological factors to explain the symptoms. Thus, the concept of "overactivity" stopped including the bladder disorders related to cancer, urolithiasis, or infections. The term "overactivity" is also no longer used in relation to neurogenic micturition disorders. For this group, the phrase "hyperreflexic bladder" is used [3,4].

Currently, both neurogenic and myogenic theories, as well as functional and structural theories, are significant in the explanation of overactivity ethiopathogenesis [5].

II. OVERACTIVE BLADDER – THE TREATMENT

Overactive bladder (OAB) is a term for a disorder whose symptoms are urinary urgency with or without incontinence, increased frequency of urination, and the need to urinate at night (nycturia).

This ailment is a problem for approximately 16% of men and women over 40 years of age. Its frequency increases to 70-80% over 80 years of age. It is the main reason for men to see a doctor because of "prostate problems". In women, this disorder is more frequently accompanied by urinary incontinence [2]. OAB symptoms suggest the overactivity of the bladder detrusor muscle, but might as well be caused by other disorders of the lower urinary tract. The most disturbing symptom is urinary urgency. It also generates other symptoms. The symptoms summary:

- Urinary incontinence – it is a dire need to urinate which is very difficult or even impossible (urgency incontinence) to resist;
- Urgency urinary incontinence – an involuntary leak of urine (of a large volume) occurring as a consequence of urinary urgency and following it directly;

- Increased frequency of urination during the day, which compels one to go to toilet more than 8 times in 24 hours;
- Nycturia – a need to wake up in the night at least once to urinate;
- Overactive detrusor – detrusor contractions confirmed by a urodynamic study occurring when the bladder is being filled during cystometry.

Overactive bladder is a clinical diagnosis formulated on the basis of symptoms, whereas detrusor overactivity (DO) is a diagnosis based on a urodynamic examination in which spontaneous contractions of the detrusor muscle are observed when the bladder is being filled. Most OAB patients have overactive detrusors, which can be both idiopathic (with no diagnosed cause) and neurogenic – related to the nerves in the urinary tract as an outcome of demyelinating diseases. The clinical diagnosis is based on the correctly collected medical history as well as on the analysis of a micturition diary kept by the patient for three days [6,7].

Treatment

Lifestyle modifications, behavioural therapy, and antimuscarinic medication pharmacotherapy are the major treatment methods. Modifying one's lifestyle and undergoing a behavioural therapy consist in the patient changing his or her hydration habits (avoiding drinking 4 hours before sleep and before journeys) and in training one's bladder, prolonging the periods before subsequent urinations. A clinically significant relief can be achieved by giving up beverages containing caffeine and alcohol and reducing the liquid intake by roughly 25%. These simple adaptation methods yield satisfying results in around 50% of patients. The treatment of more intense forms involves antimuscarinic medications (darifenacin, solifenacin, tolterodine, fesoterodine, trospium, oxybutynin and propiverine). The vasopressin analogue, desmopressin, can also be applied to decrease the nocturnal urine production. In the most difficult cases where there is no reaction to pharmacological treatment, the botulinum toxin injection to the detrusor muscle via cystoscope [8,9,10].

III. DETRUSOR UNDERACTIVITY – THE TREATMENT

Detrusor underactivity (DU) manifests through micturition and post-micturition disorders and is a risk factor for recurrent urinary tract infections and urinary retention. The causes of detrusor underactivity are multifarious; among others, they are ageing, bladder outlet obstructions (BOO), neurological diseases and autonomic nervous system disorders. The frequency of this pathology remains unknown. Collecting urological medial history is not sensitive enough, and the diagnosis is based on a urodynamic test.

Detrusor underactivity (DU) is defined by ICS (International Commision on Stratigraphy) as a reduced force or duration of detrusor contraction which impacts prolonged or partial emptying of the bladder in the normal time. The term "bladder underactivity" is often used as a synonym to detrusor underactivity, but it is not according to the ICS definitions [1-3].

The phrase "detrusor contractility disorders", defined as "decreased isometric detrusor pressure or/and the speed of urine flow in case of the absence of bladder outlet obstruction" is also used interchangeably with "detrusor underactivity", and yet they are not the same. This is because the term "contractility disorders" pertains to the functioning of the detrusor muscle, whereas detrusor underactivity is more of a collection of all symptoms that includes all causes impacting the emptying of the bladder.

The causes of detrusor underactivity can be divided into [2,3]:

- nervous background
- muscle background
- idiopathic

The causes with the nervous background – the neurogenic causes of detrusor underactivity – can be divided into metabolism-related disorders, central nervous system disorders, peripheral nervous system disorders, and infection-related disorders.

A basic metabolic cause whose complications might lead to an underactive detrusor is diabetes. Diabetic patients develop a set of symptoms related to bladder that is referred to as diabetic cystopathy – it is constituted by the loss of bladder sensitivity and underactive detrusor (one failing to contract). Diabetes leads to polyneuropathy caused by changes to glucose metabolism resulting from the generation of oxygen free radicals and oxidative stress. Both the input and output bladder tract is damaged. The disability of the axonal transport of nerve growth factor (NGF) in the input tract impacts the bladder sensitivity negatively. The demyelination, axonal degeneration, and partial autonomous denervation impact the output tract and cause changes to the bladder contractibility. The damage to small blood vessels may contribute to the decline in the

functioning of the nerves as well. The frequency of detrusor underactivity in diabetic patients is estimated to be between 23-78% in different studies [11].

The disorders to the central nervous system are usually related to multiple sclerosis (SM). SM is a chronic and a rapidly progressing disease which consists in the demyelisation of the central nervous system with deteriorating neurological deficit being the consequence. If the changes affect the cervical section of the spinal cord, 50–80% of patients are diagnosed with overactive bladder (OAB) and/or detrusor sphincter dyssynergia (DSD) in a urodynamic test.

However, some changes can affect the cervical-thoracic section of the spinal cord and in that case, around 30% of patients suffer from underactive detrusor or non-contractile detrusor.

In case of Parkinson's, the gradual degeneration of central nervous system nuclei neurons leads to neurotransmitter (dopamine) deficiency, which results in muscle trembling and stiffness and slowed down movement. Most patients suffering from this ailment are overactive, and yet 16% suffer from underactive or non-contractile detrusor.

During surgeries to the pelvic areas, parasympathetic, sympathetic, and somatic nerves might get damaged, causing detrusor underactivity. The surgeries most often related to the iatrogenic mechanism of DU development are: abdominoperineal rectum resection and radical hysterectomy. Pelvis fractures and spinal disc hernias causing a cauda equina syndrome may also potentially cause DU, even though the frequency of that remain unknown.

Sometimes even infection can cause DU – e.g. AIDS, nervous system syphilis, zoster, HSV infection, Guillain-Barre syndrome or Lyme disease [5,6,12].

IV. STRESS INCONTINENCE – THE TREATMENT

Stress incontinence is defined as involuntary urine leak caused by the increased pressure in the stomach occurring because of a physical strain (during cough, climbing stairs, lifting heavy objects). It is one of the most common causes for women to seek medical attention. This ailment is very infrequent in men, for whom it occurs most often as an outcome of prostate surgeries. Stress incontinence in women is often accompanied by pelvic floor statics disorders, which mask the occurrence of stress incontinence [5].

Therefore, after treating coleoptosia or uterovaginal prolapse, one should always apply the cough test. It has to be emphasised that some patients experience mixed types of incontinence: urgency incontinence (involuntary leak of a large volume of urine, which follows the sensation of urgency) and stress incontinence.

Stress incontinence is caused by the urethral sphincter underactivity, which, in turn, implies the possibility of conservative treatment by behavioural techniques and pelvic floor muscle exercise. An important phase of the therapeutic treatment is the avoidance of diuretic medications, controlling chronic cough and constipation, the treatment of urinary tract infections, and body weight reduction in cases of significant overweight. One has to mention also a new therapeutic option – duloxetine. However, only an insignificant percentage of female patients are completely cured after treatment with this drug. Conservative treatment can be an effective option only if the stress incontinence is a less intense one. If the symptoms are more intensified, the only right way to go is a surgery – a Burch colposuspension and mid-urethral slings fixed under the centre part of urethra via retropubic access or obturator foramina [3,5,8,12].

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